Program of Medicine Studies

MODULE
CIRCULATION

Second Year
Fourth Semester

Faculty of Medicine
Kaunas University of Medicine
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1. General information

**Supervisor of the module:** prof. Edgaras Stankevičius, Department of Physiology (edgaras@gmail.com)

**Coordinator of the module:** assoc. prof. Genuvaitė Civinskienė, Department of Physiology (genuvaite.civinskiene@med.kmu.lt; civgen78@gmail.com)

**Subjects and responsible persons:**

- Human Anatomy (assoc. Prof. Vidmantas Aželis, tel.: 327238, e-mail: vidmantas.azelis@med.kmu.lt)
- Human Histology and Embryology (prof. Angelija Valančiūtė, e-mail: angval@kmu.lt, assoc. prof. Ingrida Balnytė, tel.: 327282, e-mail: balnyte@itc.kmu.lt)
- Biochemistry (assoc. prof. Ramunė Morkūnienė, tel.: 327367, e-mail: ramunemo@delfi.lt)
- Physiology (prof. Edgaras Stankevičius, tel.: 327257, e-mail: edgaras@gmail.com, assoc. prof. Genuvaitė Civinskienė, tel.: 396053, e-mail: genuvaite.civinskiene@med.kmu.lt; civgen78@gmail.com)
- Pathological Physiology (lect. Dalia Akramienė, tel.: 327285, e-mail: dalia.akramiene@lsmuni.lt)
- Pathological Anatomy (prof. Dalia Pangonytė e-mail: dalia.pangonyte@med.kmu.lt, prof. Romualdas Gailys, tel.: 327013, e-mail: patanat@kmu.lt)
- Pharmacology (assoc. prof. Arvydas Milašius, assoc. prof. Rugilė Pilvinienė, tel.: 327242, e-mail: rugile.pilviniene@gmail.com)
- Essentials of Medical Diagnosis (lect. Daiva Emilija Rėkienė, tel.: 306093, e-mail: daiva.rekiene@gmail.com)
- General Surgery (prof. Donatas Venskutonis, tel.: 306066, e-mail: donas@medi.lt)
2. General content of the module

By analyzing the problems of this module the students gain new knowledge and apply it to the following domains:

- Morphology and physiology of the cardiovascular system;
- Development of the cardiovascular system and fetal blood circulation;
- Stoppage of the bleeding (primary hemostasis) and blood clotting (secondary hemostasis), principles of treatment of blood loss;
- Metabolism of lipoproteins and the peculiarities of the metabolism in the heart;
- Pathology of the cardiovascular system (most common) and pathophysiological mechanisms;
- Fundamentals of drug pharmacology affecting the cardiovascular system;
- Essentials of clinical evaluation of the cardiovascular system.

3. Aim and objectives of the module

The student after have studied this module should know how to define, analyze, explain and relate phenomena to the cases analyzed in the module. Attaining this aim, students must gain knowledge about the structure, function and disorders in pathological conditions, mechanisms and principles of their examination and treatment:

- heart as a pump;
- conducting system of the heart;
- coronary circulation;
- arterial circulation;
- microcirculation;
- lymphatic circulation;
- venous circulation;
- primary hemostasis and blood clotting;
- transport of oxygen (O₂) and carbon dioxide (CO₂);
- fetal blood circulation.
4. Tutorials

4.1. Case 1. Breathless artist

The artist J.D. is 34 years old. When he was 19 years old, during medical inspection for the military service, the aortic valve insufficiency was found. From this time he did not feel any signs that could limit his physical activity. However, 6 months ago he has noticed that during active physical exercise (basketball) he became breathless for a longer than usually time. The breathlessness after each physical exercise became stronger and stronger. During medical examination, it was found that the diastolic murmur could be best heard in the second intercostal space at the right side of the sternum and the systolic murmur at the apex of the heart. After the chest X-ray examination, it was found that the size of the heart has significantly increased as compared with the chest X-ray picture of J.D. two years ago. During echocardioscopic examination, a high degree of aortic regurgitation and a decrease in cardiac ejection fraction down to 35% (normal 55-75%) were found.

Medical doctor has proposed J.D. to have a cardiac operation in order to change the aortic valve. J.D. has agreed and an artificial aortic valve was implanted. During first hours after the cardiac surgery, dopamine was injected and after 6 hours following the cardiac operation J.D. cardiac output has increased from 3 L/min to 5 L/min.

*Please, explain the course and signs of the disease.*

*What is the mechanism of action of dopamine?*
Concept of the problem: heart as a mechanical pump.
Clinical symptoms: dyspnea and cardiac dysfunction.

Aim
To know the structure of the heart and to understand the cardiac mechanical activity and regulation, morphological and clinical signs of heart valve insufficiency (regurgitation), mechanisms of action of inotropic drugs and their effect on electromechanical coupling.

Learning objectives and contents
To complete an analysis of this problem the students must know:
- Development of the heart and developmental defects, structure of the heart wall.

Subject – Human Histology and Embryology
Department of Histology and Embryology
References:

Supplementary readings:

- Anatomy of heart valves and heart chambers.

Subject – Human Anatomy
Institute of Anatomy
References:
• Peculiarities of the action and electromechanical coupling in the myocardium, mechanical activity of the heart and cardiac cycle, pressure-volume changes during the cardiac cycle, preload and afterload, cardiac output and cardiac index, ejection fraction, work of the heart, origin of the heart sounds and their origin, phonocardiography, intracardiac and extracardiac regulatory mechanisms of the heart, factors affecting the cardiac output.

**Subject – Physiology**

**Department of Physiology**

**References:**


**Supplementary readings:**


• Effects of increased preload and afterload on mechanical function of the heart, pathophysiological mechanisms of dyspnea and heart failure.

**Subject – Pathological Physiology**

**Institute of Physiology and Pharmacology**

**References:**


• Morphological changes of the damaged valves and pathological anatomy of heart hypertrophy and failure.

**Subject – Pathological Anatomy**

**Clinic of Pathological Anatomy**
References:

- Mechanisms of action of inotropic drugs.

Subject – Pharmacology
Department of Basic and Clinical Pharmacology
References:

- Changes of heart sounds, characteristics of heart murmurs, principles of echocardiography, changes of arterial blood pressure in aortic valve disease.

Subject – Essentials of Medical Diagnostics
Clinic of Internal Diseases
References:

Supplementary readings:
4.2. Case 2. Rapid heart beating

In the evening, 31 years old schoolmistress S.T. during correction of the test of her pupils drank 3 cups of coffee and felt bad. She had the feeling of rapid heart beating and pulsation of blood vessels in the neck.

On the next day, the bad feeling has not changed and she went to the doctor. During medical examination, it was found that the arterial pulse was weak, frequent, and rhythmical, 180 beats/min. The increased pulsation was found by palpation of the veins in the neck. On the electrocardiogram (ECG) P waves were negative before every normal QRS complex, RR intervals were even and short. The medical doctor did the short compression in the region of the carotid sinus on the right side of the neck. However, the heart rate did not become normal. Then the doctor prescribed a fast bolus injection of adenosine intravenously. After the bolus injection of adenosine, the heart rate became normal and a normal sinus rhythm was recorded on the ECG.

*Please, explain the origin and mechanisms of disorders.*

*What are the mechanisms of action of carotid sinus compression and bolus injection of adenosine?*
Concept of the problem: cardiac rhythm and impulse propagation in the heart.
Clinical symptoms: tachycardia.

Aim
To understand the automaticity of the heart and regulation, propagation of electrical impulses in the heart, the origin of the electrocardiogram (ECG) and to know the mechanisms, clinical signs and diagnostic principles of cardiac arrhythmias, action of antiarrhythmic drugs.

Learning objectives and contents
To complete an analysis of this problem the students must know:

- Anatomy of the conducting system of the heart.

Subject – Human Anatomy
Institute of Anatomy

References:

- Cardiac automaticity, ionic mechanism of cardiac pacemaker potential, propagation of electrical impulse in the heart, nervous and humoral regulation of the cardiac electrical activity: chronotropic and dromotropic effects, the origin of the electrocardiogram (ECG), unipolar and bipolar leads of the ECG, vectorcardiography, electrical axis of the heart.

Subject – Physiology
Department of Physiology

References:


Supplementary readings:
Pathophysiological mechanisms of cardiac arrhythmias.

**Subject – Pathological Physiology**

**Institute of Physiology and Pharmacology**

**References:**


Mechanisms of action of antiarrhythmic drugs.

**Subject – Pharmacology**

**Department of Basic and Clinical Pharmacology**

**References:**


Clinical signs and ECG changes in cardiac arrhythmia.

**Subject – Essentials of Medical Diagnostics**

**Clinic of Internal Diseases**

**References:**


**Supplementary readings:**
4.3. Case 3. Lamely smoker

The patient C.I. is 60 years old and he smokes nearly 40 years 1 pack of cigarettes per day on the average. Recently he felt the pain in the right ankle and was forced to stop after 50 meters of the walk. The pain became quiet during the still standing and C.I. was able to continue walking again for about 50 meters. He complained that in the nighttime the pain in the right ankle and toes of the right feed awakened him several times. However, the pain did not become quiet when the patient C.I. sat in the bed and took the legs down.

During medical examination, the doctor found that pulsations of \textit{a. tibialis posterior} and \textit{a. dorsalis pedis} in the left leg were decreased. In the right leg it was impossible to feel the pulsations of \textit{a. dorsalis pedis}. Arterial systolic pressure in the brachial artery was 160 mmHg, in \textit{a. tibialis posterior} of the left leg was 90 mmHg, and in \textit{a. tibialis posterior} of the right leg was 60 mmHg. Blood test: total cholesterol 6,8 mmol/l (normal < 5,2 mmol/l), high density lipoprotein (HDL) cholesterol 0,8 mmol/l (normal > 1,0 mmol/l).

\textit{Please, explain the course and signs of the disease.}

\textit{What do you suggest for the treatment of the patient C.I.?}
**Concept of the problem:** peripheral arterial circulation and atherosclerosis.

**Clinical symptoms:** claudicatio intermittens, cramps of the legs.

**Aim**

To know the microstructure and anatomy of peripheral arteries and to understand the regulation of arterial circulation and disturbances of the regulation, metabolism of lipoproteins and their role in the pathogenesis of atherosclerosis, morphological signs and sequences, clinical signs and diagnostic principles of peripheral arterial disease.

**Learning objectives and contents**

*To complete an analysis of this problem the students must know:*

- Anatomy of arteries in the legs.

  **Subject – Human Anatomy**

  **Institute of Anatomy**

**References:**


- Microstructure of the arteries and arterioles.

  **Subject – Human Histology and Embryology**

  **Department of Histology and Embryology**

**References:**


**Supplementary readings:**

Metabolism of lipoproteins and their role in the formation of atherosclerosis.

Subject – Biochemistry

Department of Biochemistry

References:

Physiological characteristics of arterial circulation, arterial pulse and velocity of arterial pulse, laminar and turbulent blood flow, regulatory mechanisms of local blood flow.

Subject – Physiology

Department of Physiology

References:

Supplementary readings:

Morphological structure of the atherosclerotic plaques, their kinds, complications and causes of death.

Subject – Pathological Anatomy
Clinic of Pathological Anatomy

References:

- Complaints in chronic limb ischemia, arterial pulse palpation, principles of vascular ultrasound investigation, clinical signs and diagnostic principles of peripheral arterial disease.

Subject – Essentials of Medical Diagnostics

Clinic of Internal Diseases

References:

Supplementary readings:


The taxi-driver M.I., 52 years old, is a smoker since the age of 20 years and he smokes about 20 cigarettes/day. The only physical exercise for M.I. after the job was to go upstairs to his flat on the second floor. One day M.I. felt the pain on the left side of the chest when he was going home upstairs. The pain disappeared after a few minutes of rest. Keeping in mind that his father died at the age of 54 years due to the heart attack, on the next day M.I. went to the doctor. The arterial blood pressure of M.I. was 145/90 mmHg, pulse rate 76 beats/min, the heart sounds were without pathological changes, and electrocardiogram (ECG) at rest was normal. The doctor has prescribed to do the veloergometer exercise test in a month.

Following a few weeks after the medical consultation, M.I. had a hot dispute with another taxi-driver and suddenly felt a strong pain on the left side of the chest. The pain spread to the left shoulder and arm. Because the pain did not disappear for 1.5 hour, the co-workers called the emergency medical service. When the ambulance arrived, the doctor found M.I. excited, sweaty, and breathless. He complained to the doctor of a strong pain in the chest. The arterial pressure of M.I. was 170/100 mmHg, pulse rate 84 beats/min. By auscultation of the heart and lungs pathological changes were not found. On the ECG the elevation of ST segment in the II and III leads was determined. The patient M.I. was immediately transported to the hospital for the urgent treatment.

Please, explain what has happened to M.I.?

Please, explain the changes on the ECG and what additional tests are needed for M.I.?
Concept of the problem: coronary circulation and atherosclerosis.
Clinical symptoms: angina pectoris, dyspnea.

Aim
To know anatomy and regulation of coronary circulation, to understand the influence of atherosclerosis on coronary circulation, metabolic peculiarities of ischemic myocardium, mechanisms of action of antianginal drugs, pathogenesis, clinical signs and diagnostic principles of myocardial infarction.

Learning objectives and contents
To complete an analysis of this problem the students must know:

- Anatomy of coronary circulation.

Subject – Human Anatomy
Institute of Anatomy

References:

- The principles and regulation of metabolism in the cardiac muscle cells, disorders of metabolism during heart ischemia, basic types and mechanisms of cell death.

Subject – Biochemistry
Department of Biochemistry

References:


3. [http://www.celldeath.de/encyclo/aporev/aporev.htm](http://www.celldeath.de/encyclo/aporev/aporev.htm)

- Functional characteristics of coronary circulation and mechanisms of regulation.

**Subject – Physiology**

**Department of Physiology**

**References:**


**Supplementary readings:**


- Pathophysiological mechanisms of myocardial ischemia and infarction.

**Subject – Pathological Physiology**

**Institute of Physiology and Pharmacology**

**References:**


- Peculiarities of atherosclerosis of the coronary arteries, local complications, inducing developing of the myocardial infarction, its stages, morphology, complications and causes of death.

**Subject – Pathological Anatomy**

**Clinic of Pathological Anatomy**

**References:**
- Characteristics and reasons of angina pectoris and myocardial infarction, evaluate health history, past history, family history, risk factors, ECG changes in acute myocardial infarction - necrosis, damage and ischemia, heart contractility abnormalities in echocardiography, angiographic changes – stenosis or occlusion, myocardial damage markers (troponin T, troponin I) elevation in unstable angina pectoris and acute myocardial infarction.

Subject – Essentials of Medical Diagnostics
Clinic of Internal Diseases

References:

Supplementary readings:

- Mechanisms of action of antianginal drugs.

Subject – Pharmacology
Department of Basic and Clinical Pharmacology

References:


4.5. Case 5. Mysterious pain

A young woman T.E., 25 years old, called the family doctor following two weeks after the birth of her first baby. She complained about increasing pain and swelling in the right calf. Last night she waked up because of a sudden pain in the chest on the right side and dyspnea. When she was 19 years old, medical doctors diagnosed pulmonary thromboembolism one month from the onset of using peroral contraceptive. Her mother died suddenly a few days after her birth. A sister of her mother, who took care of T.E., had complaints about ulcers her legs. During medical inspection of T.E., the doctor found that the right calf was cyanotic and swollen. The measurement with the tape showed that the diameter of the right calf was bigger than that of the left one. The doctor urgently sent T.E. to the hospital for the treatment.

*Please, explain the complaints of T.E. and the course of the disease.*

*What treatment do you suggest for T.E?*
Concept of the problem: venous circulation and thromboembolism.
Clinical symptoms: leg and chest pain, dyspnea.

Aim
To know the anatomy and physiology of venous circulation, to understand the microcirculation and hemostasis, clinical signs and diagnostic principles of venous circulation abnormalities, pulmonary thromboembolism.

Learning objectives and contents
To complete an analysis of this problem the students must know:

- Anatomy of venous circulatory system.

  Subject – Human Anatomy
  Institute of Anatomy
  References:

- Physiological characteristics of venous circulation and regulatory mechanisms, mechanisms of blood clotting.

  Subject – Physiology
  Department of Physiology
  References:

  Supplementary readings:

- Pathophysiological mechanisms of alterations in microcirculation.
Subject – Pathological Physiology
Institute of Physiology and Pharmacology
References:

- Thrombosis in deep veins of the lower extremity, its causative factors, clinciomorphological peculiarities of thromboembolism, pathology of right heart failure.

Subject – Pathological Anatomy
Clinic of Pathological Anatomy
References:

- Clinical signs and diagnostic principles of venous circulation abnormalities, pulmonary thromboembolism.

Subject – Essentials of Medical Diagnostics
Clinic of Internal Diseases
References:

Supplementary readings:
4.6. Case 6. Fatigue, fatigue

Ms H.F., 72 years old, knows that for about 30 years her arterial blood pressure is increased, however, she used medicines irregularly. During last few months, she complained about increased weakness and fatigue in addition to increased dyspnea. Recently the weather was hot and Ms H.F. could not sleep because of breathlessness, however, she did not feel any pain in the chest.

Next day she called the doctor because of very expressed fatigue and weakness. The arterial blood pressure in the brachial artery was found to be 180/115 mmHg, and pulse rate 130 beats/min. The gallop sounds and systolic murmur in the mitral valve projection place on the chest were heard on auscultation. Moist rales over the lung bases were heard on auscultation. Edema of the legs was found by inspection. The lower margin of the liver was found about 5 cm below the costal arch by palpation of the abdomen. ECG showed the electrical signs characteristic of left ventricular hypertrophy. Echocardi oscopy showed that the ejection fraction was 30%. The chest X-ray showed changes characteristic of the left-sided heart failure.

*Please, explain the course of the disease and reasons of the fatigue for Ms H.F.*

*What tests and treatment do you suggest?*
**Concept of the problem:** regulation of arterial blood pressure and chronic heart failure.

**Clinical symptoms:** arterial hypertension, chronic heart failure.

**Aim**

To know the regulation of arterial blood pressure and pathophysiological mechanisms of arterial blood pressure regulation alterations, to understand the morphological and clinical signs, complications, diagnostic principles of arterial hypertension, morphological and clinical signs, diagnostic principles of chronic heart failure, and mechanisms of action of arterial blood pressure lowering drugs.

**Learning objectives and contents**

*To complete an analysis of this problem the students must know:*

- Regulatory mechanisms of arterial blood pressure.

**Subject – Physiology**

**Department of Physiology**

**References:**


**Supplementary readings:**


- Pathophysiological mechanisms of arterial blood pressure regulation alterations.

**Subject – Pathological Physiology**

**Institute of Physiology and Pharmacology**

**References:**
Primary and secondary hypertension, its mechanism of developing, peculiarities of systemic damage of the arterioles, morphological damage of the internal organs, complications and causes of death.

**Subject – Pathological Anatomy**

**Clinic of Pathological Anatomy**

**References:**

Mechanisms of action of arterial blood pressure lowering drugs.

**Subject – Pharmacology**

**Department of Basic and Clinical Pharmacology**

**References:**

Clinical signs, complications, diagnostic principles of arterial hypertension, the classification of arterial hypertension, clinical signs, diagnostic principles of chronic heart failure.

**Subject – Essentials of Medical Diagnostics**

**Clinic of Internal Diseases**

**References:**

**Supplementary readings:**
5. Lectures

5.1. Development and peculiarities of the structure of the heart and blood vessels (2 hrs)

Department of Histology and Embryology
In charge – assoc. prof. I. Balnytė, professor A. Valančiūtė

Description

5.2. Electrical and mechanical activity of the heart (3 hrs)

Institute of Physiology and Pharmacology
In charge – lect. I. Korotkich (Physiology), lect. D. Akramienė (Pathological Physiology)

Description

5.3 Antiarrhythmic drugs (2 hrs)

Department of Basic and Clinical Pharmacology
In charge - lect. V. Liukaitis, assoc. prof. R. Pilviniene
Description

Antiarrhythmic drugs. Drug classification. Mechanism of action of each drug class, triggered pharmacological effect are discussed. Information about the properties of the most important representatives of separate classes of drugs, indications and side effects are presented.

5.4. Heart metabolism in normal and ischemic conditions (2 hrs)

Department of Biochemistry

In charge – assoc. prof. R. Morkūnienė

Description


References:

5.5. Coronary circulation and regulation of local blood flow. Pathophysiological mechanisms of myocardial ischemia and infarction (3 hrs)

Institute of Physiology and Pharmacology
**In charge** – lect. A. Laukevičienė (Physiology), lect. D. Akramienė (Pathological Physiology)

**Description**

5.6. **Introduction to patient clinical examination. Primary and secondary arterial hypertension. The main clinical morphological syndromes of cardiovascular system. Functional syndromes (acute and chronic heart failure)** (2 hrs)

Clinic of Internal Diseases
**In charge** – lect. D. E. Rėkienė

5.7. **Regulation of the fluidity of blood and pathological anatomy of hemostasis** (2 hrs)

Clinic of Pathological Anatomy
**In charge** – prof. R.Gailys, prof. V.Lesauskaitė

**Description**
Biological essence of thrombosis and mechanisms of its formation, morphology, outcome, significance; peculiarities in separate parts of blood circulation, disseminated intravascular coagulation (DIC). Thromboembolism, its peculiarities, morphology, and significance. Hemorrhage, its mechanisms, morphology, compensatory mechanisms, outcome, and significance.
5.8. Drugs used in heart failure (2 hrs)

Department of Basic and Clinical Pharmacology

In charge - lect. V. Liukaitis, assoc. prof. R. Pilvinienë

Description

Dopamine, dobutamine; cardiac glycosides; inhibitors of phosphodiesterase. Other drugs used in congestive heart failure (angiotensin antagonists). Mechanism of action of each drug group, site of effect and triggered pharmacological effect are discussed. Information about the properties of the most important representatives of separate classes of drugs, indications and side effects are presented.

5.9. Types, causes and consequences of bleeding. Temporary and definitive hemostasis. Hemorrhagic shock (2 hrs)

Clinic of general Surgery

In charge – prof. D. Venskutonis

Description


5.10. Pathological anatomy of the heart failure and insufficiency of peripheral blood circulation (2 hrs)

Clinic of Pathological Anatomy

In charge – prof. R.Gailys, prof. V.Lesauskaité

Description

The causes, pathogenetic and compensatory mechanisms, morphology of left-sided, right-sided and total heart failure. Ischemic factors, compensatory mechanisms,
morphology; kinds of the infarctions, its morphology, outcome, and results, peculiarities, and significance of the infarctions in different parts of the arterial system.

5.11. Arterial blood pressure regulatory mechanisms and their alterations (3 hrs)

Institute of Physiology and Pharmacology
In charge – lect. A. Laukevičienė (Physiology), lect. D. Akrāmiņē (Pathological Physiology)
Description

5.12. Hypertension disease and symptomatic hypertension. Atherosclerosis (2 hrs)

Clinic of Pathological Anatomy
In charge – prof. R.Gailys, prof. V.Lesauskaitė
Description

5.13. Drugs used in hypertension. Drugs used in the treatment of angina pectoris (2 hrs)

Department of Basic and Clinical Pharmacology
In charge - lect. V. Liukaitis, assoc. prof. R. Pilvinienė

Description
Nitrates, beta-blockers, calcium blockers; sympatoplegic-blockers of alpha and beta receptors; vasodilators. Mechanism of action of each drug group, site of effects and triggered pharmacological effects are discussed. Information about the properties of the most important representatives of separate classes of drugs, indications and side effects are presented.

5.14. Thrombosis of deep vein. Pulmonary thromboembolism. Understanding about peripheral arterial disease. (2 hrs)

Clinic of Internal Diseases
In charge - prof. A. Naudžiūnas, lect. E. Kalinauskienė

Description
Thrombosis of deep vein: reasons, clinical signs, and diagnostic principles. Pulmonary thromboembolism. Understanding about peripheral arterial disease: reasons, clinical signs, and diagnostic principles.

5.15. ECG disorders of automatism and excitability. Heart conduction abnormalities. Rhythm disturbances of mixed genesis. Enlargement and hypertrophy of heart chambers. ECG changes in ischaemic heart disease (2 hrs)

Clinic of Internal Diseases
In charge – lect. L. Jankauskienė

5.16. Understanding of ischaemic heart disease: stable angina pectoris unstable angina pectoris, myocardial infarction. Causes, clinical signs, diagnostic principles. Understanding of peripheral arterial disease (2 hrs)

Clinic of Internal Diseases
In charge – assoc. prof. P. Leišytė
6. Practicals

6.1. Microstructure of the heart wall (2 hrs)

Department of Histology and Embryology

Description

Aim:
1. To learn and understand the structure of the heart wall.
2. To find out the structure of the conducting system elements of the heart.
3. To find out consistent patterns of the heart development.

Histological micropreparations:

**Myocardium.** Using high magnification recognize and draw longitudinal section of the striated cardiac muscle cells – cardiomyocytes. These cells are connected one to other end-to-end by junctional complex – intercalated discs. Distinguish intercalated discs: they are black-staining transverse lines that cross the chains of cardiac cells at irregular intervals. Cardiac cells can attach to the neighboring cardiac cells side-to-side and they form fibers’ network. Oval shape nucleus is in the centre of the cardiac myocyte.

**Wall of the heart.** Using lower magnification recognize the layers of the heart wall: epicardium, myocardium and endocardium.

Using high magnification distinguish and draw endocardium (it is composed of a single layer of squamous endothelium and subendothelial layer of loose connective tissue). Recognize myocardium: cardiac myocytes and between them septum of the connective tissue with blood vessels, lymphatics and nerves can be found in this layer. Find and draw epicardium, which is composed of: mesothelium (it covers epicardium from the surface) and subepicardial layer (it consists of loose connective tissue with blood vessels, nerves and adipose cells).

**Atrioventricular valve of the heart.** Using lower magnification look at the surfaces of the valve: the atrial surface is plane and ventricular surface – rough. Roughness of the ventricular surface is explained by the fact that tendons of papillary muscles insert into the valve. The atrioventricular valves are attached to the annuli fibrosi, the connective tissue of which extends into valve to form its core.
The valve is covered on both sides by the endocardium (it is thicker on the ventricular side). Scattered smooth myocytes are present on the atrial side of the valve, while on the ventricular side elastic fibers are prominent. Using high magnification distinguish endothelium, subendothelial connective tissue (in the atrial surface it is thicker than in the ventricular surface) on the atrial surface, and subendothelial connective tissue with elastic and collagen fibers – on the ventricular surface.

**Conducting system of the heart (Purkinje cells).** Using lower magnification recognize Purkinje cells of the heart conducting system in the subendocardial layer. These cells are bigger than usual (contractile) cardiac myocytes, they have one or two nuclei (which can be located eccentrically in the cell), rich in mitochondria and glycogen. The myofibrils are sparse and restricted to the periphery of the cytoplasm.

Using high magnification distinguish Purkinje cells, that are pale-pink, packed in bundles and covered by connective tissue.

**References:**

**Supplementary readings:**
www.mc.vanderbilt.edu/histology/labmanual2002/labsection2/Cardiovascular03.htm
www.siumed.edu/~dking2/crr/cvguide.htm
www.portfolio.mvm.ed.ac.uk/studentwebs/session1/group51/embryology.htm
www.cellbio.emory.edu/courses/medi510/Lecture09.doc

**6.2. Electrical activity of the heart (4 hrs)**

**Department of Physiology**

**Description**
The electrocardiogram (ECG) recorded at rest. Distinguish between bipolar and unipolar leads. Designate the waves in the different leads (for example, II, V₁, V₄).
Define the parameters of the ECG (P wave, PQ interval, QRS complex, ST segment, T wave, QT interval, amplitude of R wave). The ECGs recorded by Cabrera circle. Use them for defining the electrical axis of the heart. Define electrical axis of the heart by using the Einthoven triangle. The vectorcardiogram. By the use of the computer program „EKG“ define the projection points of the vectorcardiogram for the waves R and S of each Einthoven (standard limb) leads.

References:

Supplementary readings:

6.3. Mechanical activity of the heart (4 hrs)

Department of Physiology

Description

The phonocardiogram. Heart sounds. Designate the 1st and 2nd heart sounds on the phonocardiogram. Mechanical events of the cardiac cycle. Using the simultaneous phonocardiogram and ECG recording, define the phases of the cardiac cycle. Define the length of systole and diastole. By the use of the computer program „EKG“ analyze hemodynamic parameters in high pressure parts in low parts of the cardiovascular system, obtained from healthy patients. Compare tyem with data obtained from patients with aortic stenosis, aortic regurgitation (insufficiency of aortic valve), mitral stenosis, and pulmonary stenosis. Distinguish various cardiac murmurs in each case.

References:
Supplementary readings:

6.4. Understanding of patient clinical examination (3 hrs)

Clinic of Internal Diseases

Description

References:

Supplementary readings:


Clinic of Internal Diseases

Description

Students must learn to interpret ECG and know abnormalities of automatism and excitability, rhythm disturbances of mixed genesis, conduction abnormalities
(sinoatrial block – second degree type I, type II; atrioventricular block – I, II, III degree; intraventricular block- right bundle branch block, left bundle branch block), enlargement and hypertrophy of heart chambers (left atrial and ventricular, right atrial and ventricular).

References:

Supplementary readings:

6.6. Anatomy of arteries of the lower limb (2 hrs)

Institute of Anatomy

Description
1. Internal iliac artery, its location and parietal branches: iliolumbar artery, lateral sacral artery, obturator artery. Vascularization regions of these branches.
2. External iliac artery, its location and branches: inferior epigastric artery and deep circumflex iliac artery. Vascularization regions of these branches.
3. Femoral artery and its branches: superficial epigastric artery, superficial circumflex iliac artery, external pudendal artery, deep femoral artery. Vascularization regions of these branches.
5. Anterior tibial artery and its branches: posterior and anterior tibial recurrent arteries, anterior medial malleolar artery, anterior lateral malleolar artery.
6. Posterior tibial artery, its branches: circumflex fibular artery, medial malleolar branches.
7. Plantar arteries, dorsalis pedis artery, their vascularization regions.
8. Collateral arteries of the lower limb.

References:

Supplementary readings:

6.7. Microstructure of the wall of the blood vessels (2 hrs)
Department of Histology and Embryology
Description
Aim: 1. To find out the peculiarities of the structure of the different size blood vessels wall.
2. To learn and understand the main periods of the development of the blood vessels.
3. To be able to distinguish the blood vessels of the various types.

Histological micropreparations:

Precapillary arteriole. Using lower and high magnification find and draw the precapillary arteriole in the field of the loose connective tissue. It is translucent and without formed elements of blood.

Using high magnification recognize the wall of the arteriole. Its wall consists of inner layer (endothelium and thin subendothelial layer), an intermediate layer (smooth muscle cells) and outer layer (several pericytes, branched processes of which
cover the surface of the arteriole and connect with surrounding loose connective tissue.

Thin elongated nuclei of the endothelial cells are located parallel and elongated nuclei of smooth muscle cells – are perpendicular to the axis of arteriole. Nuclei of the pericytes are rounded and bigger than the nuclei of the endothelial and smooth muscle cells.

In this micropreparation you can find blood capillaries also. Their wall is made of endothelium, basal lamina, and pericytes.

Muscular artery. Using lower magnification find the artery. Using lower and high magnification distinguish and draw layers of the wall of the artery: inner (tunica intima) – the thinnest, an intermediate (tunica media) – the thickest, and outer (tunica adventitia).

Tunica intima consists of endothelium, thin subendothelial connective tissue and wavy internal elastic lamina. An internal elastic lamina is the outermost layer of the tunica intima and separates this layer from tunica media. Tunica media consists of smooth myocytes and collagen and elastic fibers between them, and external elastic lamina (if it is visible). An external elastic lamina is the last component of the media (separates media and tunica adventitia) and is present only in the larger muscular arteries (it is more difficult to distinguish this lamina in lower magnification). Tunica adventitia consists of the connective tissue. Lymphatic capillaries, vasa vasorum and nerves also can be found in the adventitia, and these structures may penetrate to the outer part of the media.

Vein. Using lower and high magnification distinguish and draw layers of the wall of the vein: inner (tunica intima) – the thinnest, an intermediate (tunica media) and outer (tunica adventitia) – the thickest. It is more difficult to distinguish the layers of the wall of the vein than those of the artery.

Endothelial cells of the tunica intima of the vein are less flat, more rounded and more prominent in the lumen than those in the artery. Subendothelial connective tissue layer contains sparse smooth muscle cells. In the tunica media you can find more collagen fibers, a little elastic fibers and a lot of smooth myocytes, oriented longitudinally and circularly. Pay attention to the tunica adventitia: there are a lot of collagen fibers (in comparison to elastic fibers), sparse smooth muscle cells and vasa vasorum also can be found.
**Aorta (H+E).** Using lower magnification recognize and draw layers of the wall of the artery: inner (*tunica intima*) – the thinnest, an intermediate (*tunica media*) – the thickest, and outer (*tunica adventitia*). Pay attention to the subendothelial layer, which is thicker than in the muscular artery.

Using high magnification distinguish and draw endothelium in the *tunica intima*. Pay attention at the size, shape and nuclei of the endothelial cells; the subendothelial layer, which is composed of thin fibers of the connective tissue, of star-like fibroblasts and smooth myocytes. In the *tunica media* find circularly oriented elastic fibers and membranes, in between them small amount of the smooth muscle cells and collagen fibers. Collagen and elastic fibers, smooth myocytes, adipose cells and *vasa vasorum* can be found in the *tunica adventitia*.

Internal and external elastic laminae are also present in the aorta, but it is difficult to distinguish them from the many other elastic laminae in the *tunica media*.

**Aorta (azan).** Distinguish elastic laminae and elastic fibers in the *tunica media* (this staining method enables us to see elastic fibers clearly). *Tunica adventitia* is blue.

**References:**

**Supplementary readings:**
www.mc.vanderbilt.edu/histology/labmanual2002/labsection2/Cardiovascular03.htm
www.siumed.edu/~dking2/crr/cvguide.htm
www.portfolio.mvm.ed.ac.uk/studentwebs/session1/group51/embryology.htm
www.cellbio.emory.edu/courses/medi510/Lecture09.doc

6.8. Arterial pulse and blood flow regulation (4 hrs)
Department of Physiology
Description
By the use of the computer program „PULS“ record the sphygmogram of the central pulse (a. carotis) and peripheral pulse (a. radialis). Describe the properties of the central and peripheral pulse. Define the duration of systole and diastole from the sphygmogram of a. carotis. Calculate the velocity of the pulse wave. Compare quantitatively the sphygmograms of the carotid artery at rest and after physical exercise.

Venous occlusion pletismography. By the use of the computer program „COGEV“ measure the blood flow in the forearm at rest, during reactive hyperemia, and active hyperemia.

References:

Supplementary readings:

6.9. Anatomy of heart and coronary circulation (2 hrs)
Institute of Anatomy
Description
1. Right coronary artery, its location and branches:
   a) branches of the 1st segment, their locations and vascularization regions;
   b) branches of the 2nd segment, their locations and vascularization regions;
   c) posterior interventricular branch, its location, branches and vascularization regions.
2. Left coronary artery, its location and branches:
   a) anterior interventricular artery, its location, branches, vascularization regions;
   b) circumflex branch, its location, branches, vascularization regions;
3. Anastomoses and revascularization of coronary arteries.
4. Cardiac veins. Their variations.
5. Cardiac walls and valves.
References:

Supplementary readings:

6.10. Anatomy of venous and lymphatic circulation (2 hrs)
Institute of Anatomy
Description
2. Internal iliac vein and its inlets: sacral veins, rectal veins, veins of bladder, prostate, uterus and vagina.
5. Cava-caval and porto-caval anastomoses of the inferior vena cava.
6. Lymphatic circulation of the lower limb: lymphatic vessels and lymph nodes

References:

Supplementary readings:


Institute of Physiology and Pharmacology

Description
Primary and secondary hemostasis. The clotting and anticlotting mechanisms. The microcirculation and Typical alterations in microcirculation:

1. intravascular alterations with aggregation of formed elements and cells and disturbances of blood rheological properties;
2. injury of endothelialocytes and blood – vessels wall;
3. extravascular alterations. Role of microcirculation alterations during atherosclerosis, arterial hypertension, diabetes mellitus, DIC (disseminated intravascular coagulation) syndrome, shock, multiple organ dysfunction syndrome.

References:


6.12. ECG changes of myocardial necrosis, damage and ischaemia (3 hrs)

Clinic of Internal Diseases
Description
Students must learn ECG changes in cases of myocardial necrosis, injury, and ischemia. They must know ECG changes of Q-wave infarction and non Q-wave infarction. The students must learn the stage of myocardial infarction on the ECG and location of myocardial infarction.

References:

Supplementary readings:

6.13. Peripheral arterial disease and deep vein thrombosis.
Pulmonary thromboembolism (3 hrs)
Description
Induction course with clinical signs and diagnostic principles of peripheral arterial disease, deep vein thrombosis, and pulmonary thromboembolism.

References:

Supplementary readings:
(3 hrs)

Clinic of Pathological Anatomy

Description

Aim: study and illustrate by macropreparations the pathogenetic mechanisms of thrombus formation, morphology, outcome, and significance. Studying macropreparations evaluate and point out peculiarities of the thromboembolism, illustrate morphology of bleeding caused by damage of blood vessel wall and increased permeability of the wall.

Electron micrographs and histological slides:

**Thrombocyte adhesion and aggregation.** Electron micrographs \((x 8500, 32000)\). Observe adherent thrombocytes to exposed collagen in the lumen of vessel (I phase); fibrous fibrin and erythrocytes attached to degranulated thrombocytes (II phase).

**Thrombus recens venae (arteriae).** Histological slide (H+E). Find thrombus forming components: pink fibrous, a scarlet homogenous mass of thrombocytes, erythrocytes and leukocytes.

**Thrombus in organisatione.** Histological slide (H+E). Observe the growing connective tissue and endothelial cells from vessel wall into the periphery of recent thrombus.

**Thrombus organisatus et recanalisatus.** Histological slide (H+E). Note that the thrombus is organized by connective tissue, formed fissures are covered with the endothelium and filled with blood and macrophages with hemosiderin could be seen in the connective tissue.
References:


Clinic of General Surgery

Description

Aim: to help the students get acquainted with:

- conception, causes, classification, consequences and Latin terminology of bleeding;
- hemostasis, mechanisms of spontaneous hemostasis, preoperative assessment of hemostasis;
- disturbances of blood coagulation, their implications on bleeding during the operation;
- syndrome of acute bleeding;
- pathogenesis and compensatory mechanisms of central hemodynamics, microcirculation and tissue metabolism disturbances during acute bleeding;
- detection of internal bleeding (clinical, laboratory and instrumental findings) and the first aid;
- acute anemia, clinical findings;
- principles of treatment of hemorrhagic shock, compensation of blood loss;
- principles of blood saving during surgical operations;
- hematomas: types, their impact and consequences;
- first aid for bleeding, ways of temporary and definitive hemostasis.

References:
6.16. Regulation of the arterial blood pressure (4 hrs)

**Department of Physiology**

**Description**
Measurement of the arterial blood pressure by palpation and auscultatory method. Using computer program „FANY“, measure the arterial blood pressure and pulse/heart rate during the orthostatic test and physical exercise. Analyze and discuss the changes of the cardiovascular parameters. Draw graphs showing changes of the arterial blood pressure and pulse/heart rate.

**References:**

**Supplementary readings:**

6.17. Pathological anatomy of the heart failure and insufficiency of the peripheral blood circulation (3 hrs)

**Clinic of Pathological Anatomy**

**Description**
Studying macropreparations, point out mostly frequent causative factors of heart failure, group them according pathogenetical mechanisms. Evaluate and mark in the notebooks macro – and micropreparations, illustrating morphological features of left-sided and right-sided heart failure.

Electron micrographs and histological slides:
**Hypertrophy of the myocyte (phase of decompensation).** Electron micrograph (x 20000). Pay attention to the ultrastructural changes of myocytes: lesser amount of mitochondrial crists, some of them disintegrated, swelling of sarcoplasm, destruction of myofibrils.

**Hyperaemia venosa acuta et oedema pulmonum.** Histological slide (H+E). Find the dilated and filled with blood interalveolar capillaries, abundant accumulation of stained-in-pink edematous fluid (transudate), mixed with some erythrocytes.

**Hyperaemia venosa chronica pulmonum.** Histological slide (H+E and reaction of Berlin blue used for revealing iron). Find some full-blooded capillaries of fibrotic interalveolar septi, a little amount of proteinous fluid in some alveoli and lots of macrophages with hemosiderin granules (siderophages).

**Hyperaemia venosa chronica hepatis.** Histological slide (H+E). Find the dilated central vein and central lobular sinusoids, with compressed and atrophic hepatocytes between them. Pay attention to the normal structures in the peripheral regions of the lobules and determine the cause.

**References:**

**6.18. Syndromes and diseases of the cardiovascular system (3 hrs)**

**Clinic of Pathological Anatomy**

**Description**
Aim: to learn and illustrate morphogenesis and morphology of atherosclerosis of different locations blood vessels, its clinicomorphological manifestations (infarctions), morphological background of arterial hypertension disease and secondary symptomatic hypertension, rheumatic endocarditis, its main complications and causes of death.

Electron micrographs and histological slides:

**Ultrastructure of atherosclerotic plaque.** Electron micrograph (x 9000). Find and draw components of the plaque.
**Rheumatic damage of the glomerular filter.** Electron micrograph (x 29 300). Find out and mark schematically immune complexes (deposits) located in the basal membrane of the glomeruli.

**Irreversible cellular damage due to recent myocardial infraction.** Electron micrograph (x 20 000). Find decayed mitochondria, myofibrils, sarcoplasmic reticulum.

**Atherosclerosis (stenosis –x%) a. coronariae cum thrombosi.** Histological slide (H+E). Find atherosclerotic plaque narrowing the lumen of artery and draw the components of this plaque: lipids, connective tissue, deposits of calcium; evaluate the percent of the lumen narrowing. Pay attention to the components of thrombus: aggregated thrombocytes, fibrin, erythrocytes and leukocytes; evaluate and point out approximate age of the thrombus.

**Infarctus myocardii recens.** Histological slide (H+E). Find the necrotic focus: cardiomyocytes without nuclei and striation, pay attention to the acidophilic sarcoplasm. Find the myocardium without damage. Pay attention to the line of demarcation between the necrotic focus and the normal myocardium. There is inflammation response with dilated vessels and neutrophilic infiltration at the margins of the infarct.

**Nephrocirrhosis arteriolosclerotica.** Histological slide (H+E). In the cortex of kidney find arterioles with thick walls, insudated by proteinous material, and the narrowed lumen, small foci of connective tissue, atrophic tubules with proteinous plugs in the lumen. Show compensatory enlarged glomeruli.

**Nodulus rheumaticcus (necrosis fibrinoides textus connectivi).** Histological slide (H+E). Find a focus of necrosis of collagen fibers insudated by fibrin and surrounded by macrophages.

**Endocarditis verrucosa recurrens.** Histological slide (H+E). Find and mark for yourself morphological changes indicating recurrent character of endocarditis – acute fresh verrucous endocarditis: thrombotic mass on the injured valvular endothelial surface and in subendothelial tissue – elements of specific immune reactions (macrophages, lymphocytes, plasmocytes).

**References:**


Clinic of Internal Diseases

Description

References:

Supplementary readings:
6.20. The main cardiovascular syndromes (arrhythmia, arterial hypertension, heart valvular diseases) (3 hrs)

Clinic of Internal Diseases

Description
The students must learn the reasons, clinical signs and ECG changes of cardiac arrhythmia and arterial hypertension.

References:

Supplementary readings:

6.21. The main functional clinical syndromes of the cardiovascular system (acute and chronic heart failure). Peripheral arterial disease (3 hrs)

Clinic of Internal Diseases

Description

References:

Supplementary readings:
7. Seminars

7.1. Antiarrythmic drugs (2 hrs)

Department of Basic and Clinical Pharmacology

Description

Drug classification. Mechanism of action of each drug class, site of effects and triggered pharmacological effects are discussed. Information about the properties of the most important representatives of separate classes of drugs, indications and side effects.

References:


Supplementary readings:


7.2. Metabolism of lipoproteins (2 hrs)

Department of Biochemistry

Description

1. Lipid transport function of lipoproteins in blood plasma.
3. Metabolism of very low density lipoproteins (VLDL) and low density lipoproteins (LDL). LDL as a source of cell cholesterol. The LDL receptors. Biochemical aspects of atherosclerosis.

References:


7.4 Drugs used in hypertension, angina pectoris and heart failure (2 hrs)

Department of Basic and Clinical Pharmacology

Description

Positive inotropic drugs. Other drugs used in congestive heart failure. Drugs used in hypertension. Drugs used in the treatment of angina pectoris. Mechanism of action of each drug group, site of effect and triggered pharmacological effect are discussed. Information about the properties of the most important representatives of separate classes of drugs, indications and side effects.

References:


Supplementary readings:

8. Examination programme

Module “CIRCULATION”

8.1. Anatomy
1. External iliac artery, femoral and popliteal arteries, their locations and vascularization regions.
2. Arteries of leg and foot, their locations and vascularization regions.
5. Coronary arteries of heart.
6. Veins of heart.
7. Lymphatic circulation of the lower limb: lymphatic vessels and lymph nodes.

8.2. Histology and Embryology
1. Structure of different diameter blood vessels’ wall and peculiarities of their development.
2. Structure of the heart wall and peculiarities of its development.
3. Heart developmental defects: their classification and causes.
4. Heart conducting system.
5. Structure of the lymphatic and peculiarities of their development.
6. Fetal blood circulation.

8.3. Biochemistry
1. Metabolism of low density lipoproteins and its disorders.
2. Peculiarities of energy metabolism of the cardiac muscle cells.
3. Changes in myocardial metabolism during ischemia.
4. Mechanism of apoptosis involving cell surface death receptors and mitochondria.

8.4. Physiology
1. Electrical activity of the heart. Ionic mechanism of cardiac pacemaker automaticity.
2. Propagation of electrical impulse in the heart. The origin of the electrocardiogram (ECG) and vectocardiogram.
3. Electromechanical coupling in the myocardium. Mechanical activity of the heart and cardiac cycle. The pressure-volume changes during the cardiac cycle.
4. Intracardiac regulatory mechanisms of the heart.
5. Extracardiac regulatory mechanisms of the heart.
6. Velocity of blood flow. Laminar and turbulent blood flow. The compliance (capacitance) and tension of vascular wall.
7. Physiological characteristics of arterial circulation. Arterial pulse, the velocity of arterial pulse and its measurement.
9. Physiological characteristics of venous circulation and regulatory mechanisms.
10. Local blood flow regulation. Myogenic, metabolic and endothelial regulatory mechanisms.
13. Rapid and short — term regulatory mechanisms of arterial blood pressure.
14. Long-term regulatory mechanisms of arterial blood pressure.
15. Functional characteristics of coronary circulation and mechanisms of regulation.
16. Primary and secondary hemostasis: mechanisms and physiological role.
17. Balance between blood clotting and fibrinolysis. Factors important for the balance.

8.5. Pathological Physiology
1. Functional unit of microcirculation. Typical disorders of microcirculation.
2. Left- and right-sided heart failure etiology and pathogenesis.
3. Intracardiac compensatory mechanisms during the changes of preload and afterload.
4. Myocardial ischemia, etiology, risk factors and pathogenesis.
5. Primary arterial hypertension, etiology and pathogenesis.

8.6. Pathological anatomy
1. Thrombosis, its definition, pathogenesis, morphology, outcomes and results.
2. Thrombosis in the chambers of the heart, arteries, and veins, its results.
3. Disseminated intravascular coagulation, its causes, pathogenesis, morphology.
4. Embolism, its definition and regularities. Sources of thromboemboli in major et minor circles of blood circulation, morphology and results.
5. Hemorrhage, its kinds, mechanisms, terminology morphology, compensatory mechanisms and results.
6. Congestive heart failure, its causes, pathogenetic mechanisms and expression of compensation.
7. Left-sided acute and chronic heart failure, its causes, and morphology.
8. Right-sided acute and chronic heart failure, its causes and morphology.
12. Infarction, its definition and causes, morphology, and outcomes.
13. Peculiarities and results of infarction in brain, heart, spleen, kidneys, intestine, and lungs.
15. Edema and insufficiency of lymphatic circulation: pathogenesis and morphology.
17. Morphology of atherosclerotic lesions, its complications. Results of atherosclerosis of aorta, cerebral, mesenteric, and renal arteries.
18. Atherosclerosis of coronary arteries, its morphology and complications.
20. Classification of primary cardiomyopathies, clinical morphological forms, complications and causes of death.
21. Common features of pathogenesis, morphology and course of systemic connective tissue diseases. Definition of rheumatic fever, its etiology, pathogenesis, morphology of affected connective tissue.
22. Cardiovascular pathology in rheumatic fever, morphology of rheumatic endocarditis, myocarditis and pericarditis, complications and causes of death.
23. Infective endocarditis, its etiology, pathogenesis, morphology, complications.

8.7. Pharmacology
1. Inotropic agents. Mechanism of action, pharmacokinetics, therapeutic uses, adverse effects.
2. Nitrates. Mechanism of action, pharmacokinetics, therapeutic uses, adverse effects.
3. Calcium channel blockers. Mechanism of action, pharmacokinetics, therapeutic uses, adverse effects.
5. Classification of antiarrhythmic agents. Quinidine, procainamide, disopyramide. Pharmacodynamics, therapeutic uses, adverse effects.
9. Other antiarrhythmic drugs (not included in the classification of antiarrhythmics): adenosine, digoxin. Pharmacodynamics, therapeutic uses, adverse effects.
10. Classification of antihypertensive drugs. Describe main effects drugs in each group. Representatives, pharmacokinetics, therapeutic uses, adverse effects.

8.8. General Surgery
1. Preoperative assessment of hemostasis.
3. Possibilities of blood saving during the operation.

8.9. Principles of Medical Diagnostic
3. Understanding about peripheral arterial disease: causes, clinical signs, diagnostic principles.
4. Understanding about primary and secondary pulmonary hypertension: causes, clinical signs, diagnostic principles.
5. Understanding about deep venous thrombosis and pulmonary thromboembolism: causes, clinical signs, diagnostic principles.
10. Aortic regurgitation: causes, clinical signs, diagnostic principles.
11. Acute left ventricular failure: pulmonary oedema. Causes, clinical signs, diagnostic principles.
12. Chronic left ventricular failure: causes, clinical signs, diagnostic principles.
13. Chronic right ventricular failure: causes, clinical signs, diagnostic principles.
14. Electrocardiographic criterions of left ventricular hypertrophy, meanings.
15. Electrocardiographic criterions of atrioventricular junctional escape rhythm and ventricular escape rhythm.
16. Electrocardiographic criterions of ventricular premature beats and atrial fibrillation.
17. Electrocardiographic criterions of supraventricular tachycardia and ventricular tachycardia.
19. Electrocardiographic criterions of q wave myocardial infarction: hyperacute, acute, subacute, old myocardial infarction.